Urodynamics of the Neurogenic Bladder

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KEYWORDS

• Urodynamics • Neurogenic bladder • Detrusor pressure
• Spinal cord injury • Myelodysplasia

HISTORICAL REVIEW

Identification of Risk Factors in Lower Urinary Tract Dysfunction Associated with Neurologic Lesions or Diseases

Spinal cord injuries

Before the 1970s and well into the1980s and beyond, risk factors associated with lower urinary tract dysfunction were not well established. The complications associated with neurogenic vesical dysfunction and obstructive uropathy were known, but the pathophysiology of these processes was not. What happened to most people with a neurogenic bladder was recognized and even expected, but knowledge of precisely why this happened was not. For example, Bors and Comarr[1] published a landmark textbook, *Neurologic Urology*, on the neurogenic bladder in 1971, which was the authoritative work in the field for many years. A good illustration of the prevailing concepts of treatment of the neurogenic bladder after spinal cord injury at the time of the book is the manner in which intermittent catheterization was used. The authors used sterile intermittent catheterization for the first 90 days after a spinal cord injury was incurred, as after that time, "90% of patients will have recovered bladder function."[1] This was based on the work on intermittent catheterization at the Stoke-Mandeville Rehabilitation Center in England. Recovery of reflex bladder function did not imply normalcy or even safety, because the cumulative risks of neurogenic urinary tract dysfunction continued to threaten most patients with spinal cord injury over time. However, the goal of short-term intermittent catheterization was to restore balanced bladder function and thus, a catheter-free state, implying an infection-free situation. Sterile intermittent catheterization was used to treat a spinal shock bladder with the expectation of recovery of reflex bladder activity. Balanced bladder function was defined by a residual urine volume 30% or less than bladder capacity. A considerable percentage of patients did briefly achieve balanced bladder function temporarily. Those who did not and most of those who did so transiently but failed to maintain that state were usually treated with indwelling catheters.

The authors noted that the progression of lower urinary tract disease was slow, and complications related to that process developed slowly in the first 5 years after spinal cord injury, then faster during the next 10 years. In parallel with the progression of lower urinary tract disease was a progressive loss of renal function. Factors identified in association with progressive urinary and renal dysfunction included chronic catheterization, vesicoureteral reflux, bladder spasticity, and urinary infection. Treatment was not specific and was largely reactive. For example, Foley catheters were used when balanced bladder function failed. These catheters were replaced by suprapubic tubes when bladder catheters failed and then by nephrostomy tubes or ileal loop diversion as the disease progressed. DeVivo and colleagues,[2] in 1993, noted that the death rate from sepsis in patients with spinal cord injury was 82 times the expected rate based on age. In addition to sepsis, the complications of neurogenic vesical dysfunction included and still include renal failure, hypertension, stone formation, incontinence, skin breakdown, tissue loss, osteomyelitis, dystrophic calcification, urethral erosion and destruction, autonomic dysreflexia, vesicoureteral reflux, and death. These complications were well known and seemingly not preventable. As an example of the

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severity of the progressive urologic disease in patients with spinal cord injury, by 10 years after injury, most were managed by catheters. In these patients, there was a very high rate of fistula and abscess formation, a 70% calculus rate, and, with time, a very high rate of renal failure and bladder malignancy.[1]

Neurogenic bladder in children

In children with myelodysplasia, despite close observation in excellent centers, 48% developed upper tract complications by age 5 years as reported by Kasabian and coworkers[3] in 1992 and Wang and coworkers[4] in 1988. Those myelodysplastic children with very high-grade vesicoureteral reflux were often treated by cutaneous loop ureterostomy to decompress the upper urinary tracts as reported by Cass and Giest[5] in 1972. That surgery did decompress the upper urinary tract but it ignored the real problem, bladder storage pressures, which were the root cause of the ureteral obstruction, dilation, vesicoureteral reflux, and renal failure. Bauer and coworkers,[6] in 1982, reported on the management of vesicoureteral reflux in myelodysplasia. They used intermittent catheterization, cutaneous vesicostomy, and antireflux surgery. They had reasonable results, but it is clear from reading the paper that the underlying problem, detrusor pressure (Pdet), was not yet recognized. Sidi and coworkers,[7] in 1986, described their experience with 58 children with myelodysplasia, 52% of whom had vesicoureteral reflux. Of these, most responded to decompressive treatment, but 12 of 14 children with high-grade reflux required an antireflux operation. The investigators noted that in some children, upper tract changes and deterioration continued despite the surgery. Unlike children with reflux in the absence of a neurogenic bladder in whom operative intervention was nearly always successful, in myelodysplastic children, Pdet is the problem: a factor not influenced by antireflux surgery. By 2002, Simforoosh and coworkers[8] reported that ureteral reimplantation was not necessary in patients with neurogenic vesical dysfunction and vesicoureteral reflux undergoing augmentation cystoplasty, because control of Pdet achieved by the surgery eliminated vesicoureteral reflux. Conversely, Momose and coworkers,[9] in 1993, reported on 2 patients with neurogenic vesical dysfunction and vesicoureteral reflux who were judged unsuitable for augmentation cystoplasty and instead were treated by Cohen-type ureteral cross-trigonal reimplantation with predictable results; the reflux persisted as did the risk. In these children, reflux is pressure-driven and not the result of poor ureteral valve function.

Late treatment of complications related to management of the neurogenic bladder

In patients with spinal cord injury, myelodysplastic children, and patients with multiple sclerosis, ileal loop diversions or cutaneous ureterostomies are done to salvage a damaged urinary tract or, occasionally, as a prophylactic measure to prevent such damage. In either case, late outcomes for these procedures in patients with neurogenic vesical dysfunction are not good.

For example, Shapiro and coworkers,[10] in 1975, noted that although creatinine clearance remained normal over a 10-year period in most of their patients treated by ileal loop diversion, complications were serious and numerous. Cass and coworkers,[11] in 1984, reported a 22-year follow-up of children treated by ileal loop diversion. They noted that deterioration in the upper urinary tracts began to occur after 10 years in addition to a high earlier complication rate. Mitchell and coworkers[12] reported on ileal stenosis as a late complication of urinary diversion resulting in a fibrotic, noncompliant loop with a risk of renal failure. This report was amplified by Simeone and coworkers[13] in 2003.They described ileal loop stenosis as an insidious process (seen years after loop formation) of loop fibrosis, which is associated with upper tract damage. This problem, known at the University of Michigan Urology Department as old loop syndrome, can only be treated by creating a new loop. From departmental experience, this problem is seen beginning at 10 years after loop formation (Konnak J, personal communication, 2009).

Another late problem is parastomal hernia formation, which can be associated with loop obstruction, ureteral and renal damage, and bleeding from the stoma, among other problems.

Repair can be equally problematic. Pastor and coworkers[14] reported on 25 patients who underwent parastomal hernia repair and noted immediate complications in 6 patients and hernia recurrence in 11 patients (44%). Wan and colleagues,[15] in 1992, reported on 71 patients with neurogenic vesical dysfunction treated for upper urinary tract stones.

Patients with catheters or ileal loop diversions had the highest rate of stone formation and the most serious problems with stones and renal deterioration. In 1994, Breza and coworkers[16] reported on 6 patients aged 7 to 22 years after ileal loop diversion, who developed serious upper tract complications and required undiversion into a continent low-pressure reservoir with resulting

stabilization of upper urinary tract function. Hetet and coworkers,[17] in 2005, reported on 246 patients who had an ileal loop diversion between January 1990 and December 2002. Mean follow-up was 24 months, and complications included fistula, evisceration, and ileus in 46 patients; medical complications resulted in 10 deaths; late urologic complications, including fistula, hernia, and peristomal hernia, occurred in 21% of patients and other urologic complications, including pyelonephritis, ureteral ileal stenosis and stones, in 21%. Although similar complications related to incontinent urinary diversion are not often reported in elderly patients undergoing ileal loop diversion as part of extirpative pelvic surgery for malignancy, these problems are time-related; as such, they are an important issue for younger patients who are diverted early on in the course of their neurologic disease, either prophylactically or as a way to deal with complications of managing their neurogenic bladder. Early prospective management guided by urodynamic testing can prevent urinary diversion.

Other methods of management of the neuropathic bladder

Through the late 70s and into the 80s, there were sporadic reports on the management of the spinal cord injury-affected bladder with a procedure to decrease outlet resistance. These procedures included external sphincterotomy, bladder neck incision, and transurethral resection of the proximal sphincter in men and women. In men, outlet procedures required condom catheters to control the resultant incontinence, but late outcomes were reasonable in the few series reporting late results.[18,19] Success was largely judged by a decrease in residual urine volumes, which was thought to be the major factor in urinary tract infection (UTI) and thus, risk. Most men with cervical spinal lesions did not achieve low residual urine volumes, and thus, repeated sphincterotomies were often required. For example, Whitmore and coworkers,[18] in 1978, described good short-term results with anteromedian sphincterotomy in spinal cord injured patients. Petri and coworkers,[19] in 1978, described bladder neck resection or incision in 86 adults with neurogenic vesical dysfunction and noted a good effect on flow, reflux, ureteral status, and infection rates. Continence is affected by these procedures , although they do reduce bladder pressure.

Movements away from prompt urinary drainage

In 1972, Lapides and colleagues[20] introduced clean intermittent catheterization as a short-term method of management of urinary retention associated with several urologic conditions. This technique would later be applied to patients with neurogenic vesical dysfunction from several causes. Lapides,[21] in 1979, thought that bladder overdistension induced bladder ischemia, thus exacerbating problems related to infection. If bladder volumes and pressures were kept low, infection would theoretically not be a problem. The early experience with intermittent catheterization in patients with retention was favorable. Although it is probably not absolutely true that simple freedom from bladder ischemia is the major advantage of intermittent catheterization, it quickly became clear that chronic bacteriuria did not produce wholesale death from sepsis in patients who managed their bladder with clean intermittent catheterization. That, in 1972 and 1979, was an astonishing revelation, because, for years, papers noted that pyelonephritis and renal failure could follow even a single catheterization. Last and coworkers,[22] in 1966, noted a relationship between urological instrumentation and bacteremia. Barton and coworkers,[23] in 1984, examined kidneys after death in patients with end-stage renal disease related to spinal cord injury and concluded that pathologic findings of amyloidosis, acute and chronic pyelonephritis, renal abscess formation, calculus disease, and pyelonephrosis were much more common in the patient population with spinal cord injury than in those without. They concluded that more effective treatment of urinary infections might help to alleviate end-stage renal disease in patients with spinal cord injury. That ideation persists today. Chronic bacterial colonization of the neurogenic bladder managed by clean intermittent catheterization or after augmentation cystoplasty is routinely treated with antibiotics, even though it is clear that eradication of chronic bacilluria in these cases never occurs.

In 1981, Kass and others[24] from the Lapides unit at Michigan University described their experience with intermittent catheterization in children with myelodysplasia. They noted that chronic bacteriuria was not a risk factor in patients without vesicoureteral reflux (VUR) or in those with low-grade VUR. Conversely, those children with high-grade reflux and chronic bacteriuria did develop progressive upper tract disease despite intermittent catheterization. The data and the concepts described in this paper indicate that its authors were not yet aware of the risk common to any neurogenic bladder, Pdet. In meningomyelocele patients, VUR is directly related to Pdet in that it is pressure-driven. Control of pressure is thus an essential element in the treatment of the

neurogenic bladder in this patient population. Given that bacteriuria is almost always present and cannot be eradicated and that rigid control of Pdet ameliorates risk, one's attention should be directed at that variable and not at a primary effort to eradicate bacteriuria, which is not possible.

Perkash[25] noted that in contrast to those managed by sphincterotomy, patients with spinal injury managed by intermittent catheterization sometimes develop what he called "silent hydronephrosis." He meant that on routine imaging, seemingly stable patients had hydronephrosis. These patients were not monitored by urodynamic testing, which illustrates that without control of Pdet, intermittent catheterization is dangerous. The same can be said about sphincterotomy or any method of management of the neurogenic bladder that does not rely on control of bladder pressure. Until recently, the most common way to control bladder pressure and incontinence was an indwelling catheter, but that had its own serious and cumulative complications.

The identification of Pdet as a risk factor in neurogenic bladder dysfunction

Probably the first single paper on Pdet as a risk factor was published in 1978 by Light and coworkers,[26] describing upper tract deterioration in children with myelodysplasia. This paper was published in the South African Journal of Surgery and was not recognized as generally applicable to all neurogenic bladder conditions until later and certainly was not applied to the myelodysplastic population until much later. In 1981, McGuire and coworkers[27] reported that upper tract damage in a large group of myelodysplastic children was related to the Pdet required to induce leakage. Not all patients with high Pdet had upper tract deterioration at the time of the report, but all children with a high detrusor leak point pressure ultimately did develop upper tract disease when followed up by McGuire and colleagues[28] in 1983. Ghoneim and coworkers,[29] in 1989, studied a group of myelodysplastic children and found that those with higher leak point pressures had low-compliance bladders and an equally high rate of upper tract deterioration. Clinical outcomes vastly improved once that association was made, and treatment by intermittent catheterization was supplemented with drug therapy to control bladder pressure, or a procedure to decrease outlet resistance was initiated as directed by pressure monitoring. Subsequently, there have been multiple papers in the literature on Pdet as the risk factor in neurogenic vesical dysfunction. Baskin and coworkers[30] used anticholinergic agents and intermittent catheterization in myelodysplastic children with good success. Any improvement in the 39% to 48% upper tract damage rate recorded in these children before the recognition that Pdet was the problem was a huge step forward. The 39% upper tract deterioration rate is that recorded from the University of Michigan population in Ann Arbor, well after intermittent catheterization had been established as a method of treatment in these patients. The 48% number comes from another study in which children were observed but not treated with intermittent catheterization. The improvement in outcome was the result of bladder pressure management, not solely the result of clean intermittent catheterization and certainly not the result of antimicrobial therapy. Patients treated by drugs and clean intermittent catheterization were at least as bacteriuric as those patients not so treated and perhaps more so. Park and coworkers,[31] in 2001, reported on their institutional experience with sphincter dilation in myelodysplastic children monitored by the effect of that procedure on the detrusor leak point pressure and bladder compliance. The initial goal was a transient reduction in leak point pressure, buying time so that the child could avoid a cutaneous vesicostomy. An unexpected benefit was a dramatic and sustained effect on bladder compliance. This study duplicated the findings reported by Bloom and coworkers[32] in 1990 on sphincter dilation in myelodysplastic patients. These 2 studies and the effect of cutaneous vesicostomy on bladder compliance, upper tract integrity, and vesicoureteral reflux confirmed the role of the outlet in the genesis of the dangerous Pdet (Connolly and colleagues,[33] 1998). Elevated urethral resistance starts a process in which compliance deteriorates and Pdets increase. Ureteral work increases as a result but reaches a limit at about 40 cm of water ambient bladder pressure, whereupon ureteral delivery of urine to the bladder ceases. This is not due to the thickness of the bladder wall but rather, to transmission of Pdet to the ureter. However, ureteral dilation and the other effects on the ureter seen by radiography are late rather than immediate changes. For example, Ozkan and coworkers,[34] in 2005, studied a group of patients with severe bladder dysfunction that led to augmentation cystoplasty. They found an elevated detrusor leak point pressure and detrusor fibrosis, with poor storage behavior being the major risk factor for upper urinary tract deterioration. Ghoneim and coworkers[35] noted in a 1990 paper that outlet obstruction associated with detrusor leak point pressure greater than 40 cm was the most important risk factor in children with neurogenic vesical dysfunction and

that early treatment predicated on urodynamic findings could prevent upper tract damage. Kaufman and colleagues,[36] in 1996, and Flood and colleagues,[37] in 1994, compared upper tract outcomes in myelodysplastic children monitored by periodic urodynamic testing with the outcome in myelodysplastic children followed by radiographic observation only. The urodynamic testing identified elevated detrusor leak point pressures and led to early treatment and prevention of upper tract damage. This was not the case in children followed by radiographic surveillance. Treatment in the radiographic group was instituted later and, in many cases, late enough so as not to prevent damage to upper tract function. Wang and coworkers[4] described a pressure management system for the neurogenic bladder in 1988, which was generally adapted with modifications by those caring for patients with neurogenic vesical dysfunction. For example, Kim and coworkers[38] reported in 1998 that they used bladder leak point pressure as the measure of success of sphincterotomy, rather than the more traditional measurement of residual urine. Perkash,[25] in 1978, suggested that voiding pressure did not always identify patients with detrusor sphincter dyssynergia who required a sphincterotomy.

That was in the early days of long-term clean intermittent catheterization, and some workers found sphincterotomy in male patients to be more effective long-term management than clean intermittent catheterization. Conversely, by the 1990s, it was clear that the effect of sphincterotomy was on Pdet; though effective, there were disadvantages to that technique, namely incontinence.

Failure of treatment of the neurogenic bladder

However improved the ability to predict upper tract changes based on urodynamic testing, there are cases in which damage has already been done or a treatment does not bring about a satisfactory reduction in bladder pressure. In such cases, continence is also a problem, along with the ongoing risk to upper tract function. For these situations, procedures to enlarge the bladder have been used successfully. Esa and coworkers,[39] in 1990, reported on 15 patients with low bladder compliance associated with various conditions that were treated by augmentation cystoplasty, with successful resolution of bladder dysfunction and incontinence and maintenance of normal upper urinary tracts. Blavais and coworkers,[40] in 2005, reported late outcomes after augmentation cystoplasty in a diverse group of patients. Mean bladder capacity increased from 166 to 522 mL and end Pdet, or Pdet at capacity, changed from 53 to 14. Flood and coworkers,[41] in 1995, reported on 122 augmentation cystoplasties done over a 10-year period. Bladder capacity increased from a mean of 108 mL to 448 mL and upper tract deterioration ceased after augmentation cystoplasty. Krishna and Gough,[42] in 1994, studied 39 children treated by augmentation cystoplasty for abnormal bladder compliance and vesicoureteral reflux. They noted that an achieved reduction in storage pressure below 20 was associated with cessation of vesicoureteral reflux without ureteral reimplantation. Even more telling are the reports of augmentation cystoplasty before renal transplantation in patients whose renal failure was the result of poor bladder function. Zaragoza and coworkers,[43] in 1993, reported excellent results with pretransplant augmentation cystoplasty in 11 patients in whom renal failure was caused by bladder dysfunction. In 2005, similar results were reported by Mendizabal and coworkers,[44] who reported on 15 patients with renal failure related to bladder dysfunction operated on between 1979 and 2003. Seven of 15 patients had an augmentation cystoplasty, others an ileal loop diversion or a vesicostomy. Graft survival rates after transplantation were similar to those in persons with normal bladder function. In 2002, Nahas and coworkers[45] reported on 21 patients with renal failure due to severe bladder dysfunction. All 21 patients had an augmentation cystoplasty, with excellent graft survival at 53 months.

Problems with augmentation cystoplasty

There is no question that augmentation cystoplasty is effective in the treatment of low-compliance or hyper-reflexic bladder. The procedure corrects vesicoureteral reflux, improves continence function, and protects the upper urinary tract, and late outcomes are excellent. On balance, the risks posed by a poorly compliant bladder justify the use of bowel segments for bladder enlargement if bladder compliance is not successfully treated by other means. There are still serious problems related to augmentation cystoplasty, including stone formation in as many as 30%; spontaneous perforation in 7% to 20%; progression of upper tract disease, which is rare; bowel obstruction and mucus production, both lifetime risks; nocturnal incontinence; electrolyte disturbance; vitamin deficiency; chronic infection; pyelonephritis; need for intermittent catheterization; and potential development of malignancy in the augmented bladder. Augmentation cystoplasties are generally effective reasonably quickly in the postoperative period. However, not all bladder augmentations are equally effective. To ensure the desired objective, reduction of Pdet to all volumes seen by the augmented bladder on a daily

basis, patients who have an augmentation cystoplasty should be monitored frequently with urodynamic studies for the first year or two after the procedure. This can only be done urodynamically by a determination of the bladder pressure achieved by a given augmented bladder to the maximum volume recovered on intermittent catheterization.

Complications related directly to augmentation

Bertschy and coworkers,[46] in 2000, reported on complications of augmentation cystoplasty in children. In a cohort of 30 patients, 21 had recurrent symptomatic infections; 5, bladder stones; 3, intestinal volvulus; and 1, severe electrolyte disturbances. Rigaud and Le Normand,[47] in 2004, noted chronic bacteriuria in a high percentage of patients and a 10% to 15% incidence of stone formation. They estimated the rate of perforation to be 10% to 15%. In the large series consisting of 122 augmentation cystoplasty patients reported by Flood and coworkers[41] in 1995, 16% had a surgical revision of the augmentation, 21% developed bladder stones, and, of these, 30% had more than one episode of stone formation. Urinary incontinence occurred in 13% and required treatment in 6 patients and pyelonephritis was reported in 11%, but this is not very often documented by specific measures and tends to be recorded when these patients are seen by other practitioners, because they are always bacteriuric. Five patients developed a bowel obstruction and 4% had spontaneous perforation. Admittedly, this was a seriously ill population before augmentation cystoplasty, but these complications are nevertheless notable. In 2005, in a rigorous study of outcome after augmentation cystoplasty in 76 patients, Blavais and coworkers,[40] noted that although they achieved good bladder capacity and very low bladder pressures, 7 patients with interstitial cystitis failed to improve, 5 had chronic diarrhea, 11 of 26 with a stoma developed incontinence or stomal stenosis, and 4% developed renal or reservoir stones.

Malignancy is another serious problem in patients with a neurogenic bladder. Although there are definite concerns about malignant tumor development after augmentation cystoplasty, in 2003 at the West Roxbury VA Spinal Cord Injury Center, Hess and coworkers[48] noted that cancer of the bladder developed 16 to 28 times more frequently in patients with neurogenic bladder dysfunction than a normal age-matched population, even in the absence of augmentation cystoplasty. Therefore, other factors are involved in tumor development in these patients.

Nevertheless, there are numerous reports of the development of malignant tumors in the bladder remnant or bowel segment used for augmentation cystoplasty. Barrington and coworkers,[49] in 1997, reported 4 adenocarcinomas arising in the bladder remnant after augmentation cystoplasty. They concluded that these were urothelial in origin. Baydar,[50] in 2005, reported the development of a signet ring carcinoma in a gastrocystoplasty. There are reports of squamous cell carcinoma, adenocarcinoma, and transitional cell carcinoma developing in augmented bladders. They include reports from more than 10 centers. The exact rate of increase in the incidence of malignancy after augmentation cystoplasty is not clear. This lack of clarity can be attributed to (1) the risk of malignant transformation in neurogenic bladders in general and (2) the not unreasonable assumption that malignancy developing after bowel augmentation is more likely to be reported than that which develops when neurogenic bladder dysfunction is managed without augmentation. This is currently an unknown. What is known is that augmentation cystoplasty tends to be done in young and even very young people.

The incubation time for malignant transformation seems to be long, and there are no well-established methods of surveillance, although periodic endoscopy and cytology have been advocated. The sensitivity and specificity of these methods has not been established (Hamid and colleagues,[51] 2003).

Other methods to control bladder pressure

Several other treatments to decrease Pdet are in use. These include autoaugmentation, electrical stimulation, vibratory stimulation, and botulinum toxin injection into the detrusor muscle or the external sphincter. Autoaugmentation was described by Cartwright and Snow[52] in 1989 and has been used in some centers with good and not-so-good results. Stohrer and coworkers,[53] in 1999, reported good results in 62 patients with spinal cord lesions from the Spinal Cord Injury Center in Murnau, Germany. MacNeilly and coworkers,[54] in 2003, reported poor long-term outcomes after autoaugmentation in children with neuropathic dysfunction.

Botulinum toxin injected into the detrusor muscle or external sphincter is effective as reported by Schurch[55] and Schmidt and coworkers[56] in 2006. The effects are transient, lasting up to 7 months, and the material is expensive.

Sacral root stimulation combined with dorsal root section has been used for many years to control Pdet and autonomic dysreflexia and to stimulate voiding. In centers with experience, results are generally excellent. In 2003, Vastenholt

and coworkers[57] reported a series of 42 patients with excellent results. There are numerous reports from Europe, Canada, and the United States with similar data; for example, Kutzenberger and colleagues[58] in 2005 reporting Deiter Sauerwein's extensive experience with some 440 cases over a 17-year period.

Vibratory stimulation intended to induce ejaculation in spinal cord-injured men had an unexpected side effect: modulation of detrusor activity that was quite profound. Laessoe and coworkers[59] reported on these effects in 2001 from Denmark. Clinical trials have started, and devices for this kind of stimulation are being tested.

To a large extent, these newer treatment methods were the result of dissatisfaction with certain aspects of augmentation cystoplasty enumerated earlier. However, all these treatments are designed to control bladder pressure, and evaluation of their efficacy is determined by urodynamic testing and in clinical outcome. Early on in the course of an injury or in the life of a child with myelodysplasia, adequate control of bladder pressure can be achieved with drugs and intermittent catheterization. It can also be achieved by reduction in outlet resistance, but that is also problematic. However, there is a link between outlet resistance and poor compliance. The effects of a reduction in outlet resistance are complex, and they are not described simply by a lower leak point pressure. In spinal cord injury patients with detrusor sphincter dyssynergia, myelodysplastic patients with elevated detrusor leak point pressures, and adult men with high-grade benign prostatic hyperplasia, there are definite and sustained improvements in bladder storage function and compliance that follow a reduction in outlet resistance. Because altered compliance is the major risk factor for upper tract damage and incontinence, this is the desired outcome. A reduction in outlet resistance cannot be said to be superior to intermittent catheterization, because the latter favors preservation of continence. There are still cases in which bladder function cannot be improved by medication, clean intermittent catheterization, and the other measures mentioned earlier, possibly because the process is so long-standing that the bladder muscles have been replaced by fibrous tissue. In these cases, bladder enlargement procedures still have value. Augmentation cystoplasty has definitely altered the natural history of neurogenic vesical dysfunction, in that it reliably controls bladder pressure. Should patients be selected by urodynamic testing for early intensive intervention, the rate of augmentation cystoplasty would probably be dramatically decreased.

Urodynamics and the neurogenic bladder

The goal of any therapy for the neurogenic bladder is to keep Pdets within very narrow limits, thus preserving upper tract integrity and continence. There are a few rules. Compliance on the bladder pressure response in incremental volume is the most important single test of risk. Compliance can be measured by a simple cystometrogram or more complex urodynamics, including the subtracted pressure system in which abdominal pressure measured with a rectal catheter is continuously subtracted from bladder pressure to provide true Pdet. This can be supplemented with an electromyographic recording of the activity of the external sphincter and video observation of the bladder, urethra, and ureters during the study to provide more information. However, for measured compliance to be meaningful, it must be determined over the volume range actually seen by the bladder studied. For example, a 33–year-old man with a T6 spinal cord injury is treated with 3 medications to control Pdet and provide continence. He catheterizes himself every 4 to 6 hours and obtains, as a maximum volume, 560 mL. He has never achieved a volume higher than 600 mL. His urodynamic testing must determine that his bladder compliance is acceptable for the entire volume range his bladder sees on a daily basis: 1 to 600 mL. Safe bladder pressures are less than 20 at all volumes. Somewhat higher pressures at terminal volumes in a given bladder are not harmful, but as a general rule, one aims for a Pdet less than 20. In another example, a 39-year-old man with multiple sclerosis is not capable of self-catheterization. He has, on initial urodynamic testing, a residual urine of 549 mL, and at 800 mL, he has a sustained high pressure detrusor contraction with detrusor sphincter dyssynergia. A Pdet of 122 cm is recorded over a 40-second time period with little or no flow. Obviously, bladder compliance in this patient from 0 to 549 mL is not important. This bladder only sees volumes from 549 to 800+ mL. Whatever the treatment, medication and clean intermittent catheterization, sphincterotomy and condom catheter drainage, or (as we chose) Botox A 100 units injected into the external sphincter, precise data on bladder capacity and mean residual urine volume is required to accurately evaluate bladder compliance.

In this case, at 1 month post-Botox, postvoid residual volume was 132 and a voiding contraction occurred at 500 mL. Compliance was slightly abnormal from 300 to capacity, but Pdet was less than 20 and voiding pressure at mid flow was 34. This is an acceptable result.

As a rule, in patients with neurogenic bladders who report stable urinary tract function, either

a compliance test or an upper tract study should be done on a yearly basis. Of these, urodynamic testing is more accurate and provides much better prognostic data.

For those patients who report a febrile or symptomatic UTI, it is imperative that a compliance study and an upper tract evaluation be done quickly. Treatment of a UTI does not solve the problem in most patients with neurogenic bladder dysfunction, and the infection only recurs unless the functional problem is identified. In most cases, the problem involves bladder pressure.

Patients who report incontinence should also be studied, because in most cases, incontinence is the result of uncontrolled bladder pressure. Most of these patients would have been treated for the incontinence with antibiotics before the urologist knows about them, so forget that as a cause. It rarely is the sole cause of any problem in this population.

Spinal cord injury

After recovery from spinal shock, a period of quite rapid evolution of bladder and urethral function occurs. Bladders progress from low-pressure compliant systems to high-pressure obstructed systems quite quickly. Regular periodic urodynamic testing is essential. Caught early, the bladder responses to volume can be controlled with medication. Once high pressures are well established, it is more difficult to reverse them and recreate a low-pressure environment. Given a chance, medication is started before reflex bladder contractility is established in the hope of preventing it entirely. Rarely, if the spinal cord injury is partial and incomplete and recovery occurs, the drugs can be stopped without harm. After 18 months to 2 years, stable bladder dysfunction is usually achieved but cannot be assumed. Moreover, bladder function continues to slowly evolve and should still be monitored.

Sphincterotomy patients

Periodic evaluation, especially in patients with cervical lesions, is mandatory on a 6-month to 1-year basis, by testing the Pdet at which urine flows from the external meatus and not by a measurement of residual urine. This is a detrusor leak point pressure, the pressure at which flow past the distal sphincter occurs, and it requires fluoroscopy, but if that is not available, watching for urine to egress from the external meatus is acceptable. If the pressure is 5 or less, all is well. If it is higher, caution and frequent testing are required. The sphincterotomy should be redone if pressures climb toward 30 or 35. A small urodynamic catheter is ideal for this and all other testing described in this article. The International Continence Society's definition of a detrusor leak point pressure is the lowest pressure in the absence of a detrusor contraction or a change in abdominal pressure (Pabd) causing urine flow—a good definition, but wrong, because in that case, there would be no flow. The detrusor leak point pressure is the Pdet at which there is urine flow past the distal sphincter. The Pdet can be a result of a phasic contraction, poor compliance, and/or fibrosis of the bladder, but the measurement is of Pdet, however produced. That definition specifically excludes Pabd as the expulsive force.

NEOBLADDER AND AUGMENTATION CYSTOPLASTY PATIENTS

These patients take time to develop capacity, and any unexplained incontinence or febrile UTI should be promptly evaluated. These are complex situations and video testing is best, because bilateral high-grade VUR may occur, which renders compliance testing useless. Where reservoir pressures are elevated, medical therapy can be used, guided by repeat urodynamics. If there is leakage from a neourethra, the cause can be determined with video urodynamics. Leakage at low reservior pressure is likely the result of a dysfunctional flap valve, whereas leakage at high reservior storage pressure is more likely related to impaired reservoir compliance.

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